1: J Mol Cell Cardiol. 1999 Jan;31(1):167-78.

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PUBLISHER FULL-TEXT ARTICLE

F1-ATP synthase beta-subunit and cytochrome c transcriptional regulation in right ventricular hemodynamic overload and hypertrophically stimulated cardiocytes.

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Cardiac hypertrophic growth secondary to hemodynamic pressure overload causes changes in energy requirements that may involve the transcriptional upregulation of oxidative phosphorylation genes. Therefore, two representative nuclear-encoded genes, the mitochondrial F1-ATP synthase beta-subunit (beta-subunit) and cytochrome c (cyt c), were examined in a feline chronic pulmonary artery banded right ventricular pressure-overload model. In the hypertrophying right ventricle, beta-subunit and cyt c mRNA levels increased after two and seven days, during the peak growth response. To examine cardiac transcriptional regulation, neonatal rat cardiac myocytes (cardiocytes) were transiently transfected with beta-subunit promoter constructs ranging from -1519 nucleotides (nt) upstream of transcription initiation as well as cyt c promoter constructs ranging from -726 nt. A full-length p1519beta-subunit/Luc construct was alpha-adrenergically inducible by 275% (+/-30%) with this activation being mapped to an enhancer region between -1519 to -1480 nt. Smaller constructs containing more proximal promoter elements were not inducible. Additionally, the full-length and enhancer deleted beta-subunit constructs were also inducible in electrically stimulated cardiocytes, suggesting a different mechanism of activation. Cyt c constructs containing known constitutive elements from -191 to -167 nt and -139 to -84 nt were responsible for the majority of the reporter activity of the full-length promoter but were not inducible in the presence of phenylephrine. Hence, we show that promoter regions containing elements common in other metabolism-related gene families are active in neonatal rat cardiocytes. Once more, we have identified a beta-subunit genomic region responsive to alpha-adrenergic and electrical stimulation.

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